ENVIRONMENTAL
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ABSTRACTS &
BIBLIOGRAPHY

July 1977

# focus: LEAD POISONING

DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE - PUBLIC HEALTH SERVICE CENTER FOR DISEASE CONTROL, ATLANTA, GEORGIA 30333

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CENTER FOR DISEASE CONTROL BUREAU OF STATE SERVICES ENVIRONMENTAL HEALTH SERVICES DIVISION

#### **FOREWORD**

Environmental Health Abstracts & Bibliography presents a survey of recently published literature in the field. Effort is made to keep the abstracts as current as possible and sufficiently informative to enable the reader to decide whether the original article would be of interest to him or her. For the benefit of the reader, where possible the address of the first author is included with each abstract. Future issues will be devoted to various other environmental health topics.

In compiling these abstracts we utilize the National Library of Medicine's interactive retrieval service, MEDLARS II. Under this system, both foreign and domestic biomedical periodicals are searched for material dealing with or related to environmental health. We also utilize the libraries of Emory University, the Center for Disease Control and other federal agencies.

Individuals desiring to be placed on the mailing key to receive *Environmental Health Abstracts & Bibliography* as published should write to the Center for Disease Control, Attention: Environmental Health Services Division, Bureau of State Services, Atlanta, Georgia 30333.

Vernon N. Houk, M.D.
Director
Environmental Health Services Division

#### **CONTENTS**

FOREWORD								. ii
GENERAL								. 1
DIAGNOSIS & SCREENING .								. 3
EPIDEMIOLOGY								.7
RESEARCH & EVALUATION								. 10
TREATMENT & PREVENTION								14

#### **GENERAL**

## SUB-CLINICAL LEAD POISONING IN CHILDREN.

Donald Baltrop—Pediatric Unit, St. Mary's Hospital Medical School, London W.2. J CHILD PSYCHOL PSYCHIATRY 17(3): 225-227, July 1976.

The discovery of a blood lead concentration greater than 40  $\mu$ g/100 ml is an indication of undue absorption and merits an investigation of potential sources, measures to prevent further exposure and a search for other similarly exposed children in the family group and in the locality. The urgency with which these steps are taken should be related to the severity of the exposure. Although only a minority of such children will require chelation therapy, it may even sometimes be necessary to consider removal of the child from the contaminated environment until the source of lead has been identified and made safe.

# PICA AND ELEVATED BLOOD LEAD LEVEL IN AUTISTIC AND ATYPICAL CHILDREN.

Donald J. Cohen, Warren T. Johnson, and Barbara K. Caparulo – 333 Cedar Street, New Haven, Connecticut 06510. AM J DIS CHILD 130(1):47-48, January 1976.

Authors' abstract: Children with severely atypical development often display pica, habitual mouthing, and odd food preferences as symptoms from the first year of life. Such children can ingest dangerous amounts of lead even in environments that are usually considered safe. Mean blood lead concentration was notably higher in 18 autistic children than in 16 nonautistic psychotic children or in ten normal siblings. Fifteen (44%) of the psychotic children (autistic and nonautistic) had blood lead levels greater than two standard deviations above the mean for normal controls. Behavioral and neurological sequelea of elevated blood lead level may be obscured in severely disorganized children. Screening for blood lead should be part of the medical care of these vulnerable children with pica.

### THE BURDEN OF LEAD: HOW MUCH IS SAFE?

M. A. Kasowski, and W. J. Kasowski-CAN MED ASSOC J 114(6): 573-574, March 20, 1976.

Various studies have raised the question of whether a blood lead level of 40  $\mu g/dl$  is indeed safe. This is important because once lead poisoning has reached the stage when clinical symptoms are observable, the prognosis is not favorable. The most recent evidence increasingly suggests that a blood level of 40  $\mu g/dl$  is too high to protect the population, particularly the very young. The United States Environmental Protection Agency (USEPA) has taken a stand, announcing at the international conference that it was lowering its definition of elevated blood lead from 40 to 35  $\mu g/dl$ .

# THE EFFECT OF CLIMATIC STIMULATION ON LEAD MOBILIZATION IN CHILDREN.

H.J. Einbrodt, E.G. Schultze, A. Schröder, and J. Rosmanith—Abt. Hygiene und Arbeitsmedizin der Med. Fakultat der RWTH, Lochnerstrabe 4-20, 5100 Aachen. OEFF GESUNDHEITSWENS 38(6): 378-382, June 1976.

English summary: "In children from a lead contaminated industrial area, it was assumed that they had formed lead depots in various tissues. The literature reports on the dangers of a lead depot formation and mobilization during changes in metabolism and in stress situations, without, however, reference to exact studies. We exposed the children with high lead burden to a change in climate (vacation at the North Sea). The blood lead levels rose rapidly in the course of the vacation as a result of the mobilization from the lead depots. An increased excretion of lead in urine could not be definitely shown. A thalasso-therapy for children with lead burden is recommended. however this requires stringent medical supervision because of the lack of demonstrable lead excretion."

## THE LEAD PROBLEM IN CHILDREN: DICTUM AND POLEMIC.

Harold Zarkowsky—Associate Professor of Pediatrics, Washington University School of Medicine, St. Louis, MO 63110. CURR PROBL PEDIATR 6(9): 1-47, July 1976.

The "lead problem" in children is a continuum of increasing absorption and retention of lead and the resultant metabolic and clinical toxicity. This includes children with minimally, moderately or extremely elevated blood lead levels; children with or without symptoms; and children with evidence of varying degrees of metabolic toxicity. While lead screening was initially set up to discover children with lead poisoning, these screening programs have also identified asymptomatic children with minimally to moderately elevated blood levels, certainly evidence of increased absorption, but not necessarily a harbinger of overt lead poisoning. This group of children numerically is the greatest concern. Within the group are children who are on their way to a steadily increasing burden of lead. The medical approach to the problem tries to select those at greatest risk, and continued research strives to determine the risk to all children with too much lead.

- Anonymous: Lead and multiple sclerosis. J R COLL GEN PRACT 26(169):622-626, Aug 1976.
- Antti-Poika M, Hassi J: Metal-induced fever. DUODECIM 92(9):457-460, 1976.
- \*Barltrop D: Sub-clinical lead poisoning in children. J CHILD PSYCHOL PSYCHIA-TRY 17(3):225-227, Jul 1976.
- Boeckx RL, Postl B, Coodin FJ: Letter: Leaded gasoline inhalation. CAN MED ASSOC J 114(11):999, Jun 5, 1976.
- \*Cohen DJ, Johnson WT, Caparulo BK: Pica and elevated blood lead level in autistic and atypical children. AM J DIS CHILD 130(1):47-48, Jan 1976.

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- \*Einbrodt HJ, Schultze EG, Schröder A, Rosmanith J: The effect of climatic stimulation on lead mobilization in children. OEFF GESUNDHEITSWES 38(6):378-382, Jun 1976.
- Gerdes N: Uptake and excretion of lead in workers in a concentrate warehouse in a mining town in Greenland. UGESKR LAEGER 138(42):2584-2586, Oct 11, 1976.
- Gliniecka M: Case of non-occupational plumbism with severe course. POL TYG LEK 31(26):1129-1130, Jun 28, 1976.
- Grandjean P: Lead problems (editorial). UGESKR LAEGER 138(42):2580, Oct 11, 1976.
- Green VA, Wise GW, Callenbach J: Lead poisoning. CLIN TOXICOL 9(1):33-51, 1976.
- Hammond PB: Proceedings: Air standards for lead and other metals. J OCCUP MED 18(5):351-355, May 1976.
- Hussey HH: Letter: Words eaten and lead poisoning. JAMA 235(9):908, Mar 1, 1976.
- \*Kasowski MA, Kasowski WJ: The burden of lead: how much is safe? CAN MED ASSOC J 114(6):573-574, Mar 20, 1976.
- Kawai M: Urinary non-precipitable lead in lead workers. BR J IND MED 33(3):187-192, Aug 1976.
- Lehnert G: Chronic heavy metal poisoning. INTERNISH (Berlin) 17(8):411-419, Aug 1976.
- \*Zarkowsky HS: The lead problem in children: dictum and polemic. CURR PROBL PEDIATR 6(9):1-47, Jul 1976.
- Zook BC, London WT, Sever JL, Sauer RM: Experimental lead paint poisoning in non-human primates. I. Clinical signs and course. J MED PRIMATOL 5(1):23-40, 1976.

#### DIAGNOSIS AND SCREENING

FREE ERYTHROCYTE PROTOPORPHY-RIN AS AN INDICATOR OF THE BIO-LOGICAL EFFECT OF LEAD IN ADULT MALES. I. RELATIONSHIP BETWEEN FREE ERYTHROCYTE PROTOPORPHY-RIN AND INDICATORS OF INTERNAL DOSE OF LEAD.

L. Alessio, P.A. Bertazzi, F. Toffoletto, and V. Foa-Clinica del Lavoro "L. Devoto", Universita di Milano, Via S. Barnaba 8, I-20122 Milano, Italy. INT ARCH OCCUP ENVIRON HEALTH 37(2):73-3, June 3, 1976.

The relationship between free erythrocyte protoporphyrin (FEP) and the indicators of an internal dose of lead (PbB, PbU, PbU-EDTA) was considered in a group of adult male subjects with varying lead exposure, whose PbB values ranged from 15-150 µg/100 ml; a highly significant correlation was found between FEP and PbB, PbU, and PbU-EDTA. The data obtained in the investigation suggests that FEP is a useful test to assess metabolic damage in adult subjects arising from an "abnormal" lead absorption and to evaluate the amount of "active deposit" of the metal present in the body.

FREE ERYTHROCYTE PROTOPORPHY-RIN AS AN INDICATOR OF THE BIO-LOGICAL EFFECT OF LEAD IN ADULT MALES. II. COMPARISON BETWEEN FREE ERYTHROCYTE PROTOPORPHY-RIN AND OTHER INDICATORS OF EF-FECT.

L. Alessio, P.A. Bertazzi, O. Monelli, and V. Foa-Clinica del Lavoro "L. Devoto" del' Universita di Milano, Via S.Barnaba, 8, I-20122 Milano, Italy. INT ARCH OCCUP ENVIRON HEALTH 37(2):89-105, June 3, 1976.

In this study the relationship existing between PbB and the other indicators of effect were examined so as to compare their behavior with that of free erythrocyte protoporphyrin (FEP) and establish whether there is a field of application where the erythrocyte metabolite is to be preferred. It was confirmed that FEP possesses good predictive validity at PbB levels of 60 and 70  $\mu$ g/100 ml; for such PbB levels, also aminolaevulinate dehydratase (ALAD), measured with the European Standardized Method, displayed high validity. Validity of the urinary metabolites was rather modest. FEP was well correlated also with other indicators, suggesting that the erythrocyte metabolite may be used to predict both dose and effect.

## ERYTHROCYTE FLUORESCENCE AND LEAD INTOXICATION.

K.G.A. Clark—Department of Haematology, Guys Hospital, London SE1. BR J IND MED 33(3):193-195, August 1976.

Author's abstract: Blood samples from people exposed to inorganic lead were examined by fluorescence microscopy for excess erythrocyte porphyrin, With continued lead absorption, fluorescent erythrocytes appeared in the circulation of workers handling this metal or its compounds, and they progressively increased in number and brilliance. These changes ensued if the blood lead concentration was maintained above 2.42  $\mu$ mol/1 (50  $\mu$ g/100 m1), and preceded any material fall in the hemoglobin value. At one factory, 62.5 percent of 81 symptomless workers showed erythrocyte fluorescence attributable to the toxic effects of lead. Excess fluorocytes were found in blood samples from a child with pica and three of her eight siblings. These four were subsequently shown to have slightly increased blood lead concentrations. Fluorescence microscopy for excess erythrocyte porphyrin is a sensitive method for the detection of chronic lead intoxication. A relatively slight increase in the blood lead is associated with demonstrable changes in erythrocyte porphyrin content. The procedure requires little blood, and may be performed upon stored samples collected for lead estimation. The results are not readily influenced by contamination, and provide good confirmatory evidence for the absorption of biochemically active lead.

MEASUREMENT OF FREE ERYTHRO-CYTE PROTOPORPHYRIN IN BLOOD COLLECTED ON FILTER PAPER AS A SCREENING TEST TO DETECT LEAD POISONING IN CHILDREN.

Bernard Davidow, George Slavin, and Sergio Piomelli—Bureau of Laboratories, The City of New York Department of Health, New York, NY 10016. ANN CLIN LAB SCI 6(3):209-213, May—June 1976.

A procedure for the measurement of free erythrocyte protoporphyrin (FEP) in a drop of blood collected on filter paper is described. The method is useful as a screening test for lead poisoning in children. Based on the FEP finding and blood lead tests, asymptomatic children are classified into four major categories. These categories reflect the degree of risk to the asymptomatic child and suggest a course of action to follow.

#### ZINC PROTOPORPHYRIN DETERMINA-TION: A RAPID SCREENING TEST FOR THE DETECTION OF LEAD POISONING.

Alf Fischbein, Josef Eisinger, and William E. Blumberg-Mount Sinai School of Medicine of The City University of New York, Fifth Avenue and 100 Street, New York, NY 10029. MT SINAI J MED NY 43(3):294-299, May-June 1976.

Community exposure resulting from a variety of products containing lead or lead compounds has rendered childhood lead poisoning a major medical problem in modern society. Protoporphyrin determination has been found to be a very useful test for the medical surveillance of lead-exposed individuals. Besides being simple and inexpensive, zinc protoporphyrin (ZPP) determination with the hematofluorometer provides a useful index of lead exposure. At present, it is probably the most practical test that reflects the actual biologic effect of lead.

# ERYTHROCYTE PORPHYRIN ANALYSIS IN THE DETECTION OF LEAD POISON-ING IN CHILDREN: EVALUATION OF FOUR MICROMETHODS.

Thomas L. Hanna, David N. Dietzler, Carl H. Smith, Santosh Gupta, and Harold S. Zarkowsky-St. Louis Children's Hospital, St. Louis, MO 63110. CLIN CHEM 22(2):161-168, February 1976.

Authors evaluated four procedures for determination of erythrocyte porphyrin: double extraction with ethyl acetate/acetic acid-HC1, single extraction with ethanol, single extraction with acetone, and direct solubilization with detergent-buffer. The ethyl acetate procedure, when used with two portions of HC1, apparently gives complete recovery of porphyrin and is suitable for reference as a comparison method. The ethanol procedure gives a high and consistent recovery and is technically simpler. The acetone procedure gives low and variable recovery of porphyrin, and the detergent-buffer method is subject to serious hemoglobin interference; neither of these two procedures offers any technical advantage. Stability of samples and methods for standardization were explored. A procedure for expressing results in terms of erythrocyte Zn-protoporphyrin content is given. Because of its stability, coproporphyrin is useful as a daily working standard. The ethyl acetate and ethanol methods are about equally efficient for detecting lead intoxication. Because of its simplicity, the ethanol method seems to be the best for use in screening.

## LEAD ENCEPHALOPATHY: CASE REPORTS.

I. Harris-Department of Paediatrics, Mdantsane Hospital, East London, CP. S AFR MED J 50(35):1371-1373, August 14, 1976.

Describes case reports of six children, all 2 years of age or younger, presenting with convulsions at a hospital pediatric unit. Five of the 6 children were anemic. In only 3 of the 6 patients was basophilic stippling present and in 2 of these it was of the fine variety which is not supposed to be diagnostic of lead poisoning. The cerebrospinal fluid showed a pleocytosis of up to 30 cells/mm and a protein of from 70 to 240 mg/100 ml (this mild albuminocytological dissociation may be helpful diagnostically). In no case was papilloedema found, presumably because the age of the children allowed splaying of the skull sutures. The first 4 patients initially were thought to have meningoencephalitis or cerebral tumors, illustrating the diagnostic difficulties which may arise in an overcrowded and overworked pediatric unit.

FEDERALLY-ASSISTED SCREENING PROJECTS FOR CHILDHOOD LEAD POISONING CONTROL – THE FIRST THREE YEARS (JULY 1972-JUNE 1975).

Donald R. Hopkins and Vernon N. Houk-Environmental Health Services Division, Center for Disease Control, Atlanta, GA 30333. AM J PUBLIC HEALTH 66(5):485-486, May 1976.

Results of the first three years of screening and follow-up under the auspices of the Federal Childhood Lead Poisoning Prevention Program indicate that a significant beginning has been made in a nationwide approach to an important public health problem. Over the three year period, 78,785 dwelling units of children with undue lead absorption were inspected for excess leadbased paint in the interior. Of the 60.9 percent found to contain lead-based paint on at least one surface accessible to a child, permanent measures to reduce the lead hazard were undertaken in 52.0 percent. Results indicate that a significant beginning has been made in efforts to control childhood lead poisoning although they do not reveal how many episodes of illness or death from lead poisoning have been averted.

# EXPERIENCE WITH A DIRECT READING DEDICATED FLUOROMETER FOR DETERMINATION OF ERYTHROCYTE PROTOPORPHYRIN.

R. Klein, P. Usher, and P. Madigan—Child-hood Lead Poisoning Prevention Program, The Commonwealth of Massachusetts Department of Health, Jamaica Plain, MA. PEDIATR RES 10(8):720-721, August 1976.

Values for erythrocyte protoporphyrin (EP) in the blood of children as determined by the direct reading fluorometer have been presented as a reference for those planning to use such instruments as they become available. Mean concentration of EP in children whose blood lead concentration was less than 30  $\mu$ g/dl was 34.7  $\mu$ g/dl whole blood  $\pm$  13.4 (SD). Mean EP concentration in children with blood lead concentrations of 40-59  $\mu$ g/dl was 80.8  $\mu$ g/dl. It was 158  $\mu$ g/dl for children with blood lead concentrations greater that 59  $\mu$ g/dl. All children in

this last group had concordant values for Pb and EP on the first or second test.

#### LP IN THE ER?

Henrietta K. Sachs. ILL MED J 149(4):365-367, April 1976.

Author's abstract: Lead poisoning programs that screen for evidence of undue lead absorption have effectively decreased morbidity and deaths by early detection and treatment of asymptomatic children. However, screening programs, where they exist at all, are directed toward the well-child population. Mothers are discouraged from bringing potentially infectious sick children to screening sessions or infant clinics. The irritable, vomiting child, with obscure signs of early lead encephalopathy, is taken not to a screening station, but to an ER where too often he receives an incorrect diagnosis and medication inappropriate for lead poisoning, only to return later with flagrant lead encephalopathy. The urgency of identifying these children at the initial hospital visit is demonstrated by examination of recent hospital and clinic records and autopsy protocols.

# DOES YOUR STATE HAVE A FREE LEAD TESTING PROGRAM FOR CHILDREN?

Ned V. Schimizzi, Associate Professor, Department of Curriculum and Instruction, State University College at Buffalo, 1300 Elmwood Avenue, Buffalo, NY 14222. J SCH HEALTH 46(6):356-357, June 1976.

Buffalo, New York has a free lead testing clinic. Children under the age of three and living in the "lead belts" of the city were tested for toxic lead levels. One third had high lead levels in their blood. Toxic lead levels in children negatively affect brain cell function and may cause brain damage and/or learning disabilities as well as hyperactivity. Pre-school children with toxic lead levels are more likely candidates for cerebral or learning disabilities, including optic atrophy. Lead encephalopathy is a cause of death. Renal, myocardial and metabolic disorders may also result from lead poisoning. The child is "de-leaded" or treated for lead toxicity by a three-step approach: (1) Possible sources of lead are removed; (2) treatment with chelating agents; and (3) neurological and psychological assessment at the time of diagnosis and during the following years is necessary for quick detection of neuro damage, behavioral deviation and for appropriate therapy.

- Ahlgren L, Liden K, Mattsson S, Tejning S: X-ray fluorescence analysis of lead in human skeleton in vivo. SCAND J WORK ENVIRON HEALTH 2(2):82-86, Jun 1976.
- \*Alessio L, Bertazzi PA, Toffoletto F, Foa V: Free erythrocyte protoporphyrin as an indicator of the biological effect of lead in adult males. I. Relationship between free erythrocyte protoporphyrin and indicators of internal dose of lead. INT ARCH OCCUP ENVIRON HEALTH 37(2):73-88, Jun 3, 1976.
- \*Alessio L, Bertazzi PA, Monelli O, Foa V: Free erythrocyte protoporphyrin as an indicator of the biological effect of lead in adult males. II. Comparison between free erythrocyte protoporphyrin and other indicators of effect. INT ARCH OCCUP ENVIRON HEALTH 37(2):89-105, Jun 3, 1976.
- \*Clark KG: Erythrocyte fluorescence and lead intoxication. BR J IND MED 33(3):193-195, Aug 1976.
- Cyran J: Symptoms and differential diagnosis of acute exogenous poisoning. INTERN-IST (Berlin) 17(8): 376-385, Aug 1976.
- \*Davidow B, Slavin G, Piomelli S: Measurement of free erythrocyte protoporphyrin in blood collected on filter paper as a screening test to detect lead poisoning in children. ANN CLIN LAB SCI 6(3):209-213, May-Jun 1976.
- \*Fischbein A, Eisinger J, Blumberg WE: Zinc protoporphyrin determination: a rapid screening test for the detection of lead poisoning. MT SINAI J MED NY 43(3):294-299, May-Jun 1976.

- \*Hanna TL, Dietzler DN, Smith CH, et al: Erythrocyte porphyrin analysis in the detection of lead poisoning in children: evaluation of four micromethods. CLIN CHEM 22(2):161-168, Feb 1976.
- \*Harris I: Lead encephalopathy: case reports. S AFR MED J 50(35):1371-1373, Aug 14, 1976.
- \*Hopkins DR, Houk VN: Federally-assisted screening projects for childhood lead poisoning control: the first three years (July 1972-June 1975). AM J PUBLIC HEALTH 66(5):485-486, May 1976.
- \*Klein R, Usher P, Madigan P: Experience with a direct reading dedicated fluorometer for determination of erythrocyte protoporphyrin. PEDIATR RES 10(8):720-721, Aug 1976.
- North AF: Screening in child care. AM FAM PHYSICIAN 13(2):85-95, Feb 1976.
- Poh-Fitzpatrick MB, Lamola AA: Direct spectrofluorometry of diluted erythrocytes and plasma: a rapid diagnostic method in primary and secondary porphyrinemias. J LAB CLIN MED 87(2):362-370, Feb 1976.
- \*Sachs HK: LP in the ER? ILL MED J 149(4):365-367, Apr 1976.
- \*Schimizzi NV: Does your state have a free lead testing program for children? J SCH HEALTH 46(6):356-357, Jun 1976.

#### **EPIDEMIOLOGY**

LEAD POISONING IN CHILDREN OF LEAD WORKERS—Home Contamination with Industrial Dust.

Edward L. Baker, Jr., David S. Folland, T.A. Taylor, Myron Frank, Wendy Peterson, George Lovejoy, Dennis Cox, Jere Housworth, and Philip J. Landrigan—Bureau of Epidemiology, Center for Disease Control, Atlanta, GA 30333. N ENGL J MED 296(5):260-261, February 3, 1977.

During December 1975, and January 1976, after an investigation of occupational lead poisoning at a secondary lead smelter in Memphis, Tennessee, 91 children and 12 wives of current and recently terminated workers were questioned. Standard techniques were used to analyze blood samples from family members for lead and erythrocyte protoporphyrin concentrations. Families of production workers having children one to six years of age with neighboring families having children of the same age range were matched; no control family member worked in a lead plant. In each pair of homes so selected, authors assessed lead content of painted surfaces by x-ray fluorescence, using a portable x-ray source detector and an attached digital analyzer. A painted surface was considered positive for leadbased paint if it contained ≥ 2 mg of lead per square centimeter. At each house, one dust sample was collected with preweighed cotton-gauze swabs by a modification of the method of Vostal et al. Authors determined lead concentration of a solution of the aciddigested sample by means of anodic stripping voltametry.

## CURRENT STATUS OF LEAD EXPOSURE AND POISONING IN CHILDREN.

J. Julian Chisolm, Jr.—Department of Pediatrics, Baltimore City Hospitals, 4940 Eastern Avenue, Baltimore, MD 21224. SOUTH MED J 69(5):529-531, May 1976.

New information indicates the very young absorb and retain a larger proportion of dietary lead than adults, that nutritional factors are important, and that subtle alterations in nervous system function can be produced experimentally with doses insufficient to cause obvious symptoms. While new environmental exposures have been discovered, old housing with its flaking, powdery, old leaded paint and putty remains the major

hazard for young children. At the investigative level, there is a clear need for better-defined sensitive neurochemical and neurophysiologic technics with which dose-effect and dose-response relationships for the effect of lead on the developing nervous system can be defined.

## LEAD POISONING YESTERDAY AND TODAY.

Af Philippe Grandjean—send reprint requests to Nils Gerdes, Birke Alle 3, DK-2660 Brondby Strand. UGESKR LAEGER 138(42):2587-2588, October 11, 1976.

English summary: "An investigation was undertaken in a lead mine in Greenland of the uptake and excretion of lead in 18 workers. Blood and urine analyses were undertaken every third week for a period of three months. Four out of 15 workers who worked under inadequate conditions of hygiene attained blood and urine concentrations which exceeded the generally accepted danger limits for development of symptoms of poisoning. Three workers who worked under good hygienic conditions (pressurized cabins and filters on tractors) had normal concentrations of lead in blood and urine. It is concluded that it is important to be familiar with the hygienic and toxicological aspects of lead mining in order to avoid lead poisoning in workers exposed to contact with lead sulphide concentrate."

EPIDEMIOLOGICAL STUDY OF LEAD LEVELS IN THE CHILD POPULATION AND THE HOUSEHOLD ENVIRONMENT IN CIUDAD JUAREZ, CHIHUAHUA, MEXICO, AS COMPARED TO A FOUNDRY AREA IN EL PASO, TEXAS.

Blanca Raquel Ordonez, Lidia Ruiz Romero, and Rufugio Mora. BOL OF SANIT PANAM 80(4):303-317, April 1976.

English summary: "A representative overall sampling of households in Ciudad Juarez, plus 100 per cent of the households in an area northwest of that city near a foundry in El Paso, Texas, were studied to determine the blood lead level of at least one child within the 1-to-9 year age range in each home and the lead content of the dust inside the house and the dirt on its yard or grounds. The occupational risk was studied, as was

the risk of lead intake from food prepared in enamel cooking utensils. Blood lead levels were found to be highest in children whose homes were closest to the foundry. There was a positive correlation between blood lead levels and the lead content of the dust in the home and the dirt in its yard or grounds. No a ssociation was established between the higher lead levels and occupational hazards or the use of enamel cooking utensils that give off heat."

## LEAD POISONING AMONG MIGRANT CHILDREN IN NEW YORK STATE.

Peter J. Smith, Dorothy M. Nelson, and Ralph E. Stewart–Lead Poisoning Control Program, New York Department of Health, Tower Building, Empire State Plaza, Albany, NY 12237. AM J PUBLIC HEALTH 66(4):383-384, April 1976.

Out of 166 dwelling units surveyed in 97 upstate New York migrant labor camps, 40 percent were found to have potentially hazardous painted surfaces, a figure much lower than comparable surveys in upstate New York high-risk urban areas. Blood samples obtained from 79 migrant children in 1972 revealed only one child with blood lead level above 40 µg per 100 ml as compared to a 21.6 percent yield from 5,733 high-risk urban area children. A second screening program in 1974 yielded only three children of 190 tested with proven blood lead levels above 40 µg per 100 ml, although seven additional children with possible elevations were lost to follow-up.

# DETECTION OF CHRONIC LEAD EXPOSURE IN CHILDREN LIVING IN PUBLIC HOUSING.

Mary L. Watkins, Nancy J. Krohn, Michael Geraldi, E. Perry Crump, and Saburo Hara—The Diagnostic and Training Laboratory, Department of Pediatrics, Meharry Medical College, Nashville, TN. J NATL MED ASSOC 68(3):182-184, May 1976.

Authors' summary: Eleven children from an old Nashville housing project were studied for increased body burden of lead. The children had hair lead levels five and onehalf times higher than the control group. Analysis of hair lead was used as a screening procedure. This technique was useful in supporting the diagnosis of chronic lead poisoning along with other laboratory data and X-ray observation.

- \*Baker EL, Folland DS, Taylor TA, et al: Lead poisoning in children of lead workers. N ENGL J MED 296(5):260-261, Feb 3, 1977.
- \*Chisolm JJ: Current status of lead exposure and poisoning in children. SOUTH MED J 69(5):529-531, May 1976.
- Corn M: Editorial: Lead poisoning in industry, 1976. AM J PUBLIC HEALTH 66(6):531-532, Jun 1976.
- Franke W, Kyrieleis C: The occupational lead poisoning as the cause of death. MED WELT 27(6): 233-235, Feb 6, 1976.
- \*Grandjean P: Lead poisoning yesterday and today. UGESKR LAEGER 138(42):2587-2588, Oct 11, 1976.
- Levine RJ, Moore RM, McIaren GD, et al: Occupational lead poisoning, animal deaths, and environmental contamination at a scrap smelter. AM J PUBLIC HEALTH 66(6):548-552, Jun 1976.
- \*Raquel Ordonez B, Ruiz Romero L, Mora IR: Epidemiological study of lead levels in the child population and the household environment in Ciudad Jaurez, Chihuahua, Mexico, as compared to a foundry area in El Paso. Texas. BOL OF SANIT PANAM 80(4):303-317, Apr 1976.
- \*Smith PJ, Nelson DM, Stewart RE: Lead poisoning among migrant children in New York State. AM J PUBLIC HEALTH 66(4):383-384, Apr 1976.
- Tola S, Hernberg S, Vesanto R: Occupational lead exposure in Finland. VI. Final report. SCAND J WORK ENVIRON HEALTH 2(2):115-127, Jun 1976.

Tola S, Karskela V: Occupational lead exposure in Finland. V. Shipyards and shipbreaking. SCAND J WORK ENVIRON HEALTH 2(1):31-36, Mar 1976.

\*Watkins ML, Krohn NJ, Geraldi M, et al: Detection of chronic lead exposure in children living in public housing. J NATL MED ASSOC 68(3):182-184, May 1976.

#### RESEARCH AND EVALUATION

THE ROLE OF LEAD IN HYPERACTIVITY.

Oliver J. David, Stanley P. Hoffman, Jeffrey Sverd, et al—State University of New York, Downstate Medical Center, Brooklyn, NY. PSYCHOPHARMACOL BULL 12(2):11-13, April 1976.

Reports a study of 13 leaded hyperactive children treated with chelating agents, seven of whom had no known cause for their hyperactivity except increased lead levels and six other children who had a probable other cause. Assessments were made during a drug-free baseline period and at the end of the treatment protocol which extended over 12 weeks. At the end of the treatment period, all but one child had significantly lower lead levels. Overall, the findings indicate that when hyperactivity in a child is not known to be caused by any of the common or known etiologies, treating the concomitant condition of a slightly increased body lead burden with chelating agents markedly ameliorates the hyperactive condition and the associated conduct disturbance.

# LEAD AND HYPERACTIVITY. BEHAVIORAL RESPONSE TO CHELATION: A PILOT STUDY.

Oliver J. David, Stanley P. Hoffman, Jeffrey Sverd, Julian Clark, and Kytja Voeller-Downstate Medical Center, State University of New York, 450 Clarkson Avenue, Box 1195, Brooklyn, New York 11203. AM J PSYCHIATRY 133(10):1155-1158, October 1976.

Authors' abstract: Lead-chelating medication was used to treat 13 hyperkinetic school children whose blood and urine lead levels were in an elevated but "nontoxic" range. Six children with histories of etiologically relevant perinatal or developmental complications showed relatively little improvement. Seven other children with unremarkable histories, and for whom a lead etiology could thus be entertained, showed marked improvement. The authors conclude that lead may play an important role in the etiology of some cases of hyperactivity; lead-chelating agents may have a major place in the treatment of hyperactivity; and the medical workup of hyperactivity should include lead level measurements and careful consideration of other possible etiological factors.

#### ATOMIC ABSORPTION SPECTRO-PHOTOMETRY ASSESSMENT OF BLOOD AND URINE LEAD. BLOOD LEAD LEVELS IN NORMAL POPULATION.

A. Garcia de Jalon, M. Sanchez Agreda, M. P. Martinez Cortes, and M. Bassecourt Serra—C. Sanitaria. S. S. Serv. Analisis Clinicos. CRTQ Calamita, 1, Zaragoza (Espana). SANGRE (Barc) 21(2):375-386, 1976.

English summary: "A semi-micromethod, based on Hessel's technique, using a 2 ml serum sample without loss of accuracy and reproducibility, was used to assess lead levels in blood and urine. Two hundred-fifty healthy individuals, residents of Zaragoza and its province, were tested. The mean values found were: 6.5 ± 3.25 mcg/100 ml for newborns (cord blood). 9.41 ± 4.40 mcg/100 ml for children, and 16.0 ± 6.44 mcg/100 for adults. These figures vary according to age and sex, and, especially, to professional activities."

# LEAD CONTENT OF TISSUES OF BABY RATS BORN OF, AND NOURISHED BY LEAD-POISONED MOTHERS.

Narayani P. Singh, Inderjit S. Thind, Leonard F. Vitale, and Metodi Pawlow— Department of Preventive Medicine and Community Health, New Jersey Medical School, 100 Bergen St., Newark, NJ ,07103. J LAB CLIN MED 87(2):273-280, February 1976.

Authors' abstract: Three groups of Wistar rats (5 males and 5 females in each group) were given zero, 100 mg., and 200 mg. Pb (NO<sub>3</sub>)<sub>2</sub> per kilogram of body weight per day for 45 days and were mated. The lead feeding was continued throughout the duration of experiments. The blood and urinary lead, blood aminolevulinic acid dehydratase activity, and free erythrocyte protoporphyrin and urinary aminolevulinic acid were measured on these rats at the beginning of the study, at the time of mating, and prior to delivery. The tissues, including kidney, liver, heart, and brain of the newborn progeny

were analyzed for lead content. The blood lead and aminolevulinic acid dehydratase were also measured (pooled blood). These tissues contained very high amounts of lead when the newborns were killed within half an hour after their birth, a very significant reduction being found after 1 day with further reduction after 7 days. Kidney and heart contained the highest amount of lead. the levels being dose-related. Three weeks after birth the lead content of tissues rose significantly but blood concentrations remained almost identical to the levels obtained 1 day after birth. There was no evidence of lead accumulation in the brain of the babies. Twelve and a half, and 50 percent newborns of rats fed 100 mg. Pb(NO<sub>3</sub>)<sub>2</sub> and 200 mg. Pb(NO<sub>3</sub>)<sub>2</sub>, respectively died during the 3 weeks. The deaths could represent the results of placental transfer of lead or the combined effects of placental and maternal milk lead burden.

STABILITY OF ERYTHROCYTE AMINO-LAEVULINATE DEHYDRATASE UNDER SEVERAL CONDITIONS OF STORAGE OF THE HEMOLYSATE FROM HUMAN BLOOD.

K. Tomokuni-Department of Public Health, Okayama University Medical School, Okayama, Japan. CLIN CHIM ACTA 56(3):547-549, June 15, 1976.

Discusses data on the loss of enzyme activity during storage of a hemolysate from whole blood, which is the enzyme solution itself. Human blood samples were taken from 10 volunteers with no history of undue lead exposure and from 24 workers occupationally exposed to lead. A hematocrit measurement was made and divided into 3 or 4 samples of equal volume. Using one sample as a control (performing the enzyme assay immediately), the other samples were stored under several conditions. Results showed that the lead-affected aminolaevulinate dehydratase (ALAD) is very stable compared to the normal ALAD in the storage of the hemolysate at 5°C.

LEAD POISONING: ASSOCIATION WITH HEMOLYTIC ANEMIA, BASOPHILIC STIPPLING, ERYTHROCYTE PYRIMIDINE 5'-NUCLEOTIADASE DEFICIENCY, AND INTRAERYTHROCYTIC ACCUMULATION OF PYRIMIDINES.

William N. Valentine, Donald E. Paglia, Kay Fink, and Glenn Madokoro—Departments of Medicine and Pathology, University of California, Center for the Health Sciences, Los Angeles, CA 90024. J CLIN INVEST 58(4):926-932, October 1976.

Authors' abstract: Lead intoxication is accompanied by an acquired deficiency of erythrocyte pyrimidine-specific, 5'-nucleotiadase. Genetically determined deficiency of this enzyme is associated with chronic hemolysis, marked basophilic stippling of erythrocytes on stained blood films, and unique intraerythrocytic accumulations of pyrimidine-containing nucleotides. The present report documents that lead-induced deficiency when sufficiently severe gives rise to findings similar to the hereditary disorder. Whereas pyrimidine-containing nucleotides are virtually absent in the erythrocytes of normal and reticulocyte-rich blood, 12% of erythrocyte nucleotides in the blood of a patient with lead intoxication contained cytidine. Nucleotidase activity was about 25% that in normal erythrocytes and 15% or less of that expected in comparable reticulocyte-rich blood. The distribution of nucleotidase activity in patient erythrocytes is unknown, and much more severe deficiency could have been present in subsets of the cell populations analyzed. The findings indicate that the hemolytic anemia and increased basophilic stippling characteristic of certain cases of lead intoxication may share a common etiology with essentially identical features of the genetically determined disorder.

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#### TREATMENT AND PREVENTION

# ROLE OF THE LABORATORY IN THE DIAGNOSIS AND TREATMENT OF LEAD POISONING.

F. Buneaux, P. Protin, M. Besson-Leaud, and P. Fabiani—Laboratoire central de Boichimie, Service du Professeur P. Fabiani, Hotel-Dieu de Paris, 1, place du Paris-Notre-Dame, 75181 Paris Cedex 04. EUR J TOXICOL ENVIRON HYG 9(3):165-170, May-June 1976.

English summary: "Alternative current polarography has been applied to specific determination of INH and acetyl-isoniazide in serum and urine. Determinations can be achieved directly on diluted urines. The method allows measure of pharmacocinetic parameters and determination of acetylation phenotype following I.V. administration of the drug."

#### MEASURES FOR THE REDUCTION OF LEAD RISK IN CHILDREN IN AN AREA BURDENED WITH HEAVY METALS.

H. J. Einbrodt, A. Schroder, J. Rosmanith, H. W. Spohr, and M. Klostermann – Abt. Hygiene und Arbeitsmedizin der Med. Fakultat der RWTH lochnerstrave 4-20, 5100 Aachen. OEFF GESUNDHEITSWES 38(4):223-225, April 1976.

English summary: "After a screening test required by the Ministerium fur Arbeit, Gesundheit und Soziales des Landes Nordrhein-Westfalen for children living in an industrial area with heavy metals melting plants, measures became necessary to exclude excessive lead burdening in children, due to a nearby slags dump of a shut-down zinc smelter. Specific measures taken and results are reported."

## PROPHYLACTIC CHELATION THERAPY FOR LEAD EXPOSURE.

John F. Finklea – National Institute for Occupational Safety and Health, Rockville, Md. JAMA 235(15):1553, April 12, 1976.

Workers exposed or potentially exposed to lead are being treated prophylactically on an outpatient basis with chelating agents. This practice is strongly opposed by the National Institute for Occupational Safety and Health (NIOSH) and the American Occupational Medical Association. This treatment, in effect, places workers in double

jeopardy by virtue of the potentially harmful effects of such long-term drug therapy combined with continued excess exposure to lead. Chelating agents, of course, have a proper place in the therapy of acute lead intoxication, but should be administered only under proper medical supervision. Physicians should not administer such treatment to workers and then send them back to their jobs when a likelihood exists that they will continue to be overexposed to lead.

# BULLETS, JOINTS, AND LEAD INTOXICATION. A REMARKABLE AND INSTRUCTIVE CASE.

Donald M. Switz, M. E. Elmorshidy, and William M. Deyerle – Box 908, Medical College of Virginia, Richmond, Virginia 23298. ARCH INTERN MED 136(8): 939-941, August 1976.

Authors' summary: A retired teamster with abdominal pain and anemia was found to have lead intoxication presumably due to an old bullet in his ankle. Most lead particles within the body need not be removed. Lead solubility characteristics exemplified by this case cause us to recommend that bullets and lead particles facing synovial spaces should be removed.

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